REMARKS

ON

EMPHYSEMA OF THE LUNGS.

By GEORGE BUDD, M.D., F.R.S.,

PHYSICIAN TO THE SEAMAN'S HOSPITAL, DREADNOUGHT.

READ DECEMBER 10TH, 1839.

Dr. Baillie was the first to describe methodically some of the principal anatomical characters of emphysema of the lungs; namely, increased volume of the lungs, absence of their natural tendency to collapse when the chest is opened after death, and dilatation of the air-cells.

The illustrious Laennec gave a much more complete description of this condition of the lungs: he also pointed out the signs by which it may be recognised during life, and engaged for it the attention of physicians, by showing that it is the most common cause of that group of distressing symptoms which is well known under the name of asthma.

Since Laennec's time, many physicians have improved our knowledge of this disease: M. Louis especially, by his accurate researches, has thrown light on its history and symptoms, and made us acquainted with its great frequency. To Dr. Stokes

also, we are indebted for valuable observations on the symptoms of emphysema: but little, however, has been added to our knowledge of its anatomical characters, which Laennec so well described. It is necessary to my present purpose to recapitulate the chief of these.

When we inspect the chest of a person who has died with extensive emphysema of the lungs, we remark that its form, instead of being flattened and depressed, as is usual after death, is more or less cylindrical; that the ribs have lost the obliquity which they naturally have in expiration, and that the depressions which commonly exist above the clavicles are wholly, or partially, effaced; in short, that the cavity of the chest is dilated—often much more so than in the most ample inspiration of healthy persons.

When the chest is opened, the lungs are found to be unnaturally voluminous, overwrapping one another, and in some instances displacing the neighbouring viscera. They do not collapse as in the natural state; sometimes, on the contrary, they protrude as the scalpel relieves them from the pressure of the parietes of the chest. They also offer great resistance to pressure, and are with difficulty deprived of the air they contain. On further examination, the increased volume of the lung is found to be owing to dilatation of the air-cells, and this alteration exists in a higher degree towards the borders of the lung, than elsewhere. When the lung is cut into, it is observed to be unusually dry and pale.

It is one of the chief objects of this paper to show that want of elasticity in the lung—in other words, absence of its natural tendency to collapse,—is the cause of many of the other anatomical characters of emphysema, and of most of the symptoms by which this affection is recognised. In order to show this more clearly, I shall first give a general idea of the act of breathing in its natural state. In natural breathing, when the expansion of the chest has attained its limit, and inspiration is complete, a quantity of air, equal to that which has been inhaled, is again expelled; chiefly, if not wholly, by means of the elasticity of the lung, which restores that organ to the volume it had before inspiration. The *muscles* and parietes of the chest do no more than follow the lung in its collapse. In every other respect they are passive. The elasticity of the lung is the main agent of expiration. That this property of the lung is more than adequate to accomplish that action is proved by the fact, that when the chest is opened after death, and the atmospheric pressure tending to compress the lung is, consequently, equal to that which tends to dilate it, there ensues a still further expulsion of air from the lung, effected by the residual elasticity of that organ—so that the only cause which, during life, prevents more complete expiration than does actually take place, is the inability of the parietes of the chest to follow further retrocession of the lung. When, therefore, the lungs have their natural tendency to collapse, the amplitude of the act of inspiration varies with

the degree to which the parietes of the chest can follow them in their retrocession; and this degree evidently depends in great measure on the obliquity of the ribs. In man different degrees of obliquity of the ribs are rarely noticed; but the sportsman is well aware of the influence of this circumstance on the speed of his horse and his dogs. In fleet animals, indeed, the obliquity of the ribs is very considerable, and is well exemplified in the deep, keely, chests of the grey-hound and the race-horse.

These points being established, I proceed to consider some of the consequences of the absence of the natural elasticity of the lung, which, as we have seen, constitutes one of the principal anatomical characters of emphysema.

One of the first effects of this condition is, that the lungs, and with them the parietes of the chest, do not collapse as they should do in expiration; the powerful muscles of inspiration are continually acting to elevate the ribs and dilate the chest, and have not their natural antagonist. The chest becomes, in consequence, permanently dilated; often beyond the limit attained in the most ample natural inspiration. It is the permanent elevation of the ribs that gives to the chest the cylindrical form, and, by raising in turn the shoulder-blades and collar-bones, that produces the high shoulders of asthmatic persons.

When this conformation of the chest is attained, its capacity cannot be much further increased by the action of the muscles, which raise the ribs. This

circumstance gives a peculiar character to the breathing of persons affected with emphysema; the ribs being permanently raised by the dilatation of the chest, the increased capacity of that cavity which takes place in inspiration is mainly effected by the diaphragm, and the respiration is abdominal. It is owing to this circumstance that the erect posture is more necessary to asthmatics than to persons affected with pleurisy or pneumonia, in whom the respiration is of equal, or even greater, frequency; and that dyspepsia, by causing flatulence and distension of the stomach, and so opposing the descent of the diaphragm, is so often the cause of a fit. The attack of the paroxysm in the night—a peculiar feature of asthma—seems to result, not from the state of sleep, but from the horizontal posture, which causes impediment to the descent of the diaphragm. The abdominal character of the breathing is still further increased by the circumstance that the portion of lung in contact with the diaphragm is not so subject to emphysema as others. This character of the breathing is very conspicuous in horses affected with emphysema, on account of the shortness of their flanks, and is well known to horse-dealers as a sign of broken-wind.

I have said that the ribs are permanently much raised: they have, therefore, little or no space to move through in inspiration, and consequently remain almost fixed. Nothing is more striking than the contrast which may be observed in a severe fit of asthma between the labouring for breath, the

active play of the nostrils, with other marks of extreme dyspnœa, and the comparative immobility of the chest.

The modification which this affection of the lungs produces on the act of coughing is also well worthy of attention. It is plain that coughing, which is nothing more than a rapid succession of sudden and forcible expirations, must be impeded in the same way as common expiration. If we observe the cough in asthma, we perceive that the parietes of the chest are little affected by it, and that it is short and interrupted; a circumstance the more distressing because the catarrh, to which persons affected with emphysema are habitually subject, is attended with a copious secretion from the bronchial membrane. The efforts of cough being ineffectual, and the irritation of the mucus remaining, the cough repeats itself in fits. Thus, in this distressing complaint, not only is less air than natural admitted to the internal surface of the lungs, but that surface is also sheathed from its action by a copious secretion which the cough is inadequate to detach: it is on account of this combination of circumstances that difficulty of breathing attains in asthma a degree which is seldom equalled in other affections. From this it follows also, that catarrh is the great enemy of persons affected with emphysema of the lungs, and that it is in this disease, especially, that we may hope for signal advantage from the effects of appropriate climate.

The preceding remarks apply chiefly to those ex-

treme cases, in which, when the chest is opened after death, the lungs protrude; in which it is, therefore, evident that, during life, expiration must have been wholly effected by pressure from without. In less advanced cases, when the emphysema has not attained a sufficient degree to add considerably to the volume of the lung, or to oppose its retrocession to that limit beyond which the parietes of the chest cannot follow it, it is evident that emphysema cannot be a powerful cause of difficulty of breathing in the way I have explained above. The truth is, that in these cases the difficulty of breathing is not great. When, however, the emphysema is partial, and the portions of lung affected have completely lost their elasticity, the explanation above given will apply perfectly to those portions. They will not collapse during expiration, and the corresponding parietes will become permanently raised. The air also which these portions contain will be very imperfectly renewed: in fact they will contribute little to the act of breathing, which will be performed almost entirely by the rest of the lungs. When a lung that is partially emphysematous is artificially inflated, the emphysematous portions increase in volume much less than the others, and, from being prominent when the inflation was begun, those portions do not exceed the level of the rest when that process is completed.

One obvious effect of the want of action in these emphysematous portions is, that the air produces little sound in entering them; a circumstance which

explains the feebleness of the respiratory murmur, which is a well-known sign of emphysema.

Not only is an insufficient quantity of air inhaled into the emphysematous portions, but also, and for the same reason, less blood than natural flows to meet it. Laennec, in his description of emphysematous lungs, makes the following statement: "The pulmonary tissue is less moist in an emphysematous than in a sound lung, and you cannot find in the former, even towards its root, any trace of serous or sanguineous congestion."

Cases the sixth and seventh, in the first volume of his work on diseases of the chest, afford such striking illustrations of this, that I may be excused for giving an abstract of them.

The sixth case is that of a man affected with asthma and slight anasarca; who, while in the hospital for the treatment of these complaints, was cut off by small pox. In the last days of his illness he experienced great difficulty of breathing.

The following is the description of his lungs. "The lungs were without adhesions. They completely filled the cavity of the chest, and seemed to be compressed by its parietes. They did not collapse in the least on the admission of the external air. Their tissue was dryer than natural: in a few points only, which were less emphysematous than the rest, and situated in the centre or towards the root of the lungs, a small quantity of serous fluid, very frothy and slightly tinged with blood, oozed from the surfaces exposed by incisions."

The seventh case is still more striking. It is that of a man who died of emphysema of the lungs with pulmonary catarrh. There was orthopnœa in the last days of his illness, and his death seemed to result from suffocation.

When the lung was cut into, there was less crepitation than usual, and neither blood nor serum oozed from its tissue; which everywhere, except towards the root of the lung, was dryer than that of the most healthy lung. The bronchi were very red, and filled with a white, tenacious mucus.

To these cases I may add the following, which came under my own notice. In the winter of 1837 a man was admitted into the Dreadnought, affected with general emphysema of the lungs and pulmonary catarrh. He died in a state of asphyxia soon after admission.

The lungs were found extremely dry and pale; there was dark blood in the large veins of the lungs; but, except from these, scarcely a drop of blood escaped when free incisions were made in all parts of the lung. There was no pneumonia; but the small bronchial tubes contained yellow puriform mucus.

These cases are very striking; for what can be more remarkable than to find paleness, dryness, and absence of congestion in the lungs of persons who have died in a state of asphyxia; the well-known and most marked effect of that condition being the greatest possible congestion of the lungs! The conclusion is, therefore, peremptory, that during life

the natural vascularity of the lungs, at least as regards the pulmonary artery, was much diminished. This restriction is necessary; since, in the cases referred to, the bronchial membrane was red and turgid. The coincidence of the pale, aneuric condition of the pulmonary tissue, with the congested state of the mucous membrane of the bronchial tubes, in the same lung, is worthy of observation, as showing an essential difference between bronchitis and pneumonia—a difference which has its origin in the different purpose and distribution of the bronchial and pulmonary arteries.

The following case proves still more decisively the truth of the foregoing considerations.

In the month of October 1837, a man was admitted into the Dreadnought, affected with cough, difficulty of breathing, and loss of voice. The soft palate had been destroyed many years before by syphilitic ulcerations, which were now quite healed. There was a large cicatrix in the pharynx. He was much emaciated. Some time after his admission he was attacked with double pneumonia, of which he soon died.

The lower lobes of both lungs were covered with a thick coating of lymph. They were hepatized throughout, except a broad rim at the edges, which was emphysematous, and was the only portion of the lower lobes not solidified. The dry, pale, and light emphysematous border contrasted strongly with the great mass of the lower lobes, which was solid, heavy, friable and granular. The upper lobe

of the left lung was very emphysematous, and perfectly dry; it was a complete puff. (There were no tubercles.) The trachea below the larynx was contracted to the size of a large quill; the cicatrix resembled that which follows a burn.

In this case the emphysema had protected a large portion of the lung from pneumonia, and this protection must have been owing to diminished vascularity. That portion of the lung did not collapse during expiration; the air within it was not changed: in fact its function was no longer performed, and, as in fœtal life, no blood flowed there to be aerated. This fact affords an illustration of a law in physiology; that the tissue by which a function is performed becomes atrophied when that function ceases or becomes less active.

Congestions and the inflammatory affections of the lungs have been well studied; but I am not aware that any anatomist has bestowed steady attention on the remarkable diminution of vascularity which takes place in those portions of lung which have become emphysematous: yet this diminution of the capillary circulation (of the pulmonary artery), often in a considerable portion of the lung, is very remarkable in an organ whose function it is to expose all the blood in the system to the action of the air.

One effect of this condition of the lung is imperfect arterialization of the blood, and, consequently, diminution of animal heat. In the patient whose case I have already mentioned, who died in the

Dreadnought, of emphysema and bronchitis, this diminution of the temperature of the body was very remarkable. During the time he lived after his admission, his nose, lips, and tongue were cold, like those of a person in cholera.

Another consequence of this diminution of the capillary system of the pulmonary artery, is obstruction to the circulation through it: whence arise dilatation of the right cavities of the heart, and the tendency to general œdema, which is so frequently met with in emphysematous persons.

I have already alluded to the occurrence of emphysema in horses, and to its being in them, the cause of the symptoms so familiar to horse-dealers by the name of broken-wind. I have at present before me an account of the state of the lungs in twenty horses, of various ages, that were killed by the knackers. In these dissections, which were made in reference to this subject, the following circumstances were remarked.

In all, except two, the lungs were more or less emphysematous. The dilatation of the cells followed exactly the same order as in man, affecting chiefly those on the borders of the lung. The naturally thin edges of the lung were by reason of this, much thickened and rounded, and very frequently presented lobular appendices, formed by groups of cells dilated to the size of swan-drops. The anterior lobe, which in horses is a long, tongue-shaped, flap of lung, was always more affected with emphysema than any other part, and it was that lobe which so often pre-

sented the lobular appendices alluded to. Large vesicles under the pleura, filled with air and communicating with the bronchi, were observed in many instances. The emphysematous portions, as in man, were pale and dry, and remarkable for their great want of vascularity. The lobes most affected were much increased in volume; but in no instance did the lungs completely fill the chest. It must not be forgotten, however, that these horses did not die of the disease.

M. Louis found lobular appendices in man, in three only of forty cases of emphysema; whereas they were present in all but three of the twenty horses examined. Most of these horses were out of mark and old; but three of them were young, and in these the affection was less advanced than in the others.

From these observations I conclude that emphysema of the lungs is very common in horses; that in them its development follows in all particulars the same order as in man; and, therefore, that the affection has probably the same cause in both.

The investigation of the causes of emphysema of the lungs in man has furnished Mr. Jackson with novel and unexpected results. Of twenty-eight persons affected with emphysema of the lungs, he found that eighteen were the offspring of parents (father or mother) affected with the same disease, and that several of these had died in its course. In some instances, the brothers and sisters of these persons were also emphysematous. On the other hand,

of fifty persons not affected with emphysema of the lungs, three only were the offspring of emphysematous parents: whence it follows that emphysema is very frequently an hereditary disease. I need not dwell on the interest of this fact as regards man, but I would point it out to the breeders of horses as one of great importance, for there can be little doubt that the disease is transmitted in the same way in the horse. It is undoubtedly owing to ignorance or disregard of this fact, that broken-wind is so common, even in young horses.

Laennec, in his examination of emphysematous lungs, being much struck with the dilatation of the air-cells, a circumstance which so readily admits a mechanical interpretation, supposed that this affection was in every case produced by some obstruction in the air-passages, which prevented the free escape of air from the lungs; and that bronchitis, by the secretion with which it is attended, is the most common cause of this obstruction. He imagined that when the bronchial tubes were obstructed with mucus, the powerful muscles of inspiration would overcome the resistance which this mucus would offer to the entrance of air into the lungs, but that the elasticity of the lung, which is the chief agent in expiration, would be inadequate to effect its expulsion; and that the cells would in consequence be permanently dilated. This explanation, which accounted in such a plausible manner for the dilatation of the cells, was admitted by all physicians, until doubts of its correctness were excited by the

discovery made by Mr. Jackson of the hereditary nature of emphysema. The accurate researches of M. Louis have confirmed these doubts. He found that emphysema often developes itself without the occurrence of pulmonary catarrh; that very frequently it is not sensibly increased by a severe attack of this malady; and that the highest degree of emphysema is met with in the upper lobe, and at the edges of the lung, while acute catarrh is most common and most severe in its lower and posterior part. He concludes from these facts, that if catarrh have any influence on the development of emphysema, this influence is slight, and but rarely exerted. His observations show also that pneumonia has no influence in producing emphysema: that persons are not unfrequently met with who have had many attacks of pneumonia, but do not exhibit any of the symptoms of emphysema, and that emphysema already existing is often not sensibly increased by an attack of pneumonia. From these circumstances he is led to reject entirely the supposition which ascribes the dilatation of the cells to a mechanical cause. Thus Laennec, considering dilatation of the cells as the essential character of emphysema, explained it by a mechanism similar to that which causes the dilatation of other hollow organs; namely, an obstacle to the free escape of their contents; and Louis, finding that in a great number of instances no such obstacle existed, in the sense in which it was understood by Laennec,

has been induced to deny that the dilatation is caused by mechanical means.

Laennec was right in supposing that dilatation of the air-cells is occasioned by an obstacle to the free escape of their contents; but he was wrong in believing this obstacle to exist generally in the bronchial tubes. Louis was correct in stating that emphysema often comes on without the previous occurrence of bronchitis; but he was, I believe, in error, when he ascribed dilatation of the air-cells to a cause different in its nature from that which produces dilatation of other organs. Dilatation of the air-cells, like dilatation of the chest, is a necessary consequence of want of elasticity of the lung. The powerful muscles of inspiration are continually acting to dilate the chest, and thence, by virtue of atmospheric pressure, the air-cells. This agency is not counteracted as it should be, by the natural elasticity of the lung; and the air-cells, as well as the cavity of the chest, are in consequence permanently dilated.

I have already shown that the other anatomical characters of emphysema, together with most of the symptoms of this disease, result from the same cause; and I am, therefore, led to consider the absence of elasticity* of the pulmonary tissue as the funda-

* Magendie ascribes the difficulty of breathing in emphysematous persons and in broken-winded horses, to want of elasticity of the lung, but he does not attribute the dilatation of the air-cells to the same cause: on the contrary, he says, "par suite de la rupture d'un certain nombre de cellules, et de la dilatation d'un certain

mental character and primary condition of emphysema of the lungs.

The preceding remarks suggest some considerations on the subject of asthma.

Asthma has been generally ascribed to constriction of the small bronchial tubes, in consequence of spasm of the circular fibres which surround them, and which have been supposed to be muscular.

There is reason, however, to believe that these fibres, like those which compose the middle coat of arteries, are not muscular; but that they are simply fibres of elastic tissue. The bronchial tubes resemble the arteries in this, that they are cylinders destined to the passage of a fluid. Now it is evident, from the property which fluids have of propagating pressure equally in all directions, that muscular fibres surrounding these cylinders would have equal effect to propel the fluid backwards and forwards; and, consequently, would tend to dilate the air-cells, as much as to expel air from the trachea. They would not, therefore, contribute much to the act of expiration. The supposition of muscular fibres in the lungs for the purpose of expiration is, besides, perfectly unnecessary; all that is required of the lungs for the accomplishment of this act is, that they should collapse readily. Now lungs not em-

nombre d'autres, le tissue de l'organe a perdu de son elasticité, et il ne reagit plus avec une energie suffisante sur l'air qui a penetré dans son parenchyme." Leçons, t. i. p. 169.

physematous,[a] collapse when the chest is opened many hours after death, (in fact until decomposition has taken place,) as completely as immediately after death; and generally to a much greater degree than the parietes of the chest can follow them. It is evident that no part of this collapse can be owing to muscular action, since, in Mammalia, muscular irritability ceases within an hour after death; and no part of it can result from atmospheric pressure, because, when the chest is opened, the atmospheric pressure acting on the external surface of the lung, and tending to compress it, must be equal to that acting within the lung, and tending to dilate it. The elasticity of the tissues which compose the lungs must, therefore, be the sole cause of their collapse, when the chest is opened many hours after death, and this elasticity is consequently more than sufficient, in healthy lungs, for the purpose of expiration. It is difficult to conceive that muscular fibres surrounding the bronchi, could in any way contribute to the act of inspiration: so that for ordinary breathing, there is no need of a muscular power in the substance of the lungs.

On the other hand, an elastic tissue is required in the bronchial tubes, as well as in the arteries, to counteract the force which acts periodically to expand them.

During inspiration, atmospheric pressure is removed from the surface of the lung by the muscles of inspiration, the air rushes into the chest, and

tends, by its expansive force, to dilate the bronchial tubes and air-cells. This tendency is counteracted in the larger bronchial tubes by cartilaginous rings, and in the smaller ones, where less strength of material is necessary, by elastic circular fibres. It is the loss of elasticity in certain portions of these tubes from disease, that is the most common cause of dilatation of the bronchi. Although the muscularity of these fibres has been long maintained, and, since the time of Cullen, has been adduced to explain the symptoms of asthma, either by supposing, with Cullen, the spasm of these fibres, or, as an eminent living physician has preferred, their paralysis, very few attempts have been made to demonstrate the existence of this muscularity. In order to satisfy my mind on this point, I performed, with the assistance of my friend Mr. Busk, the following experiments.

A rabbit, between two and three months old, was killed by a smart blow behind the ears. As soon as its struggles were over, the trachea was taken out, and the anterior part of the cartilaginous rings removed by the scissors, in order that any motion produced by the transverse fibres at its posterior part might be more readily seen. When a portion of the trachea, thus prepared, was placed on a plate, not the slightest movement could be seen in it, nor could any be excited by the wires of a galvanic battery. One of the lungs was then removed, and placed on the plate, between two and three minutes after the struggles of the animal had ceased. The end of the bronchi in which the lung terminated, was

obstructed by light froth. No motion could be observed in this froth, or in the lung, before or after the wires were applied to different points on the surface of the lung, neither could any motion be perceived, when the lung was cut into, and the extremities of the wires were placed near one of the bronchial tubes.

The abdomen was opened at the end of five minutes, when the muscular fibres of the stomach and intestines were seen to contract slowly, but very distinctly, under the galvanic influence. At the end of ten minutes, these contractions were no longer perceptible; but vigorous contractions could still be excited in the heart, and in the muscles of the larynx.

Another rabbit, of the same age as the former, was killed in the same manner; one of the lungs was taken out as quickly as possible, and placed on the plate. Not the slightest movement could be observed in it, nor could any be excited by placing the wires of the battery at different points of its surface, or in contact with the bronchial tubes. The trachea was then removed, and treated as in the former experiment, and with the same result. The abdomen was next opened, the intestines were moving from peristaltic action. The muscles, both of the intestines and of the stomach, which was distended, contracted very distinctly when galvanism was applied. At the end of ten minutes from the death of the animal, these contractions were no longer discernible; but more than half an hour after,

contractions could be excited in the heart, and in the muscles of the larynx.

The galvanic trough employed contained fifty pairs of copper and zinc plates. The solution used was a mixture of muriatic acid and water. The chemical action of the battery was powerful, causing ecchymosed spots on the surface of the lung at the points to which the wires were applied; and opaque white spots, from coagulation of albumen, at those points of the peritoneum which were touched by the wires.

Among the older authors who made experiments to ascertain the muscularity of the bronchial tubes, Varnier is most frequently quoted. His experiments consisted in injecting chemical irritants, chiefly the mineral acids, into them. The constriction which he noticed in the small bronchial tubes as a consequence of this injection is attributable to chemical changes produced in the texture of those tubes, and cannot be adduced as proof of their muscularity.

In recent times, Wedemeyer has made experiments for the same purpose. I have been unable to refer to the work in which he has recorded these experiments, but the following is the account given of one of them by Müller. "Wedemeyer laid bare the trachea in a living dog, and freed it from cellular tissue for the space of two inches: he then cut out a portion in front, and irritated the posterior wall of the trachea mechanically and by galvanism, but could not produce the slightest contraction. Wedemeyer now opened the thorax quickly, and

removed the lungs with their bronchi. He made several sections of the larger bronchi, but could discover no sign of contractility in them. On applying galvanism, however, to the smaller branches of about one line in diameter he thought he saw them undergo a distinct contraction, but it took place very slowly." This experiment of Wedemeyer, as far as the trachea and larger bronchi are concerned, agrees with those that I have before related: and together they seem to establish that no contractions can be excited in those tubes by galvanic influence. This point, if admitted, affords an almost conclusive argument against the muscularity of the smaller tubes. For the transverse fibres in the smaller tubes have the same arrangement as in the larger, and we cannot suppose them to be of different nature without admitting a break in the law of continuity. The resemblance of the transverse fibres of the bronchial tubes to the muscular fibres of organic life—the chief argument in favour of the muscularity of the former—is certainly more striking for the fibres in the larger than for those in the smaller bronchi. The contraction witnessed by Wedemeyer in tubes of a line in diameter, resulted in all probability from chemical changes, especially the coagulation of albumen, caused by the galvanic influence. Such changes were very manifest in my own experiments. The manner in which I performed the experiment, by placing the wires, not in contact with one of the small bronchial tubes, but at different points on the surface of the lung, affords

a much more delicate test of the muscularity of the bronchi. If these were muscular, a great number of them would be excited at once when the wires were placed on the surface of the lung and the galvanic influence diffused through its mass: and their combined effect would be visible in movements of the surface of the lung, or of the froth obstructing the orifice of the terminal bronchus.

I might conclude, then, from the preceding observations and experiments alone, that the transverse fibres of the bronchi have been wrongly supposed to be muscular. But it can be shown from other and independent considerations, that these fibres are not muscles, performing a part in the ordinary acts of breathing. Supposing them to be muscular, it is evident from their arrangement and microscopic characters, that they belong to the muscles of organic life, or that they are involuntary muscles. But all the external muscles of respiration are voluntary muscles. Hence we should have engaged to accomplish the act of breathing, a voluntary and an involuntary power. The function would be easily performed as long as these powers acted in unison—that is, as long as the involuntary muscles contracted only during expiration. But, by varying the rapidity of our breathing, we should soon have the two powers opposed to each other—the involuntary muscles acting to close the bronchial tubes, while the voluntary muscles acted to expand them. We should then be able to dilate the chest only when we adjusted the inspiratory movements to the

actions of the involuntary muscles. But we perceive no necessity for such an adjustment. The test, which this circumstance affords us, is one of extreme delicacy. For, if the two powers were not *exactly* in unison, there would occur intervals, like the *beats* in music, when they would coincide or be opposed to each other. The inspiratory acts would be alternately easy and difficult, according as the voluntary and involuntary muscles were in the same or in opposite phases. But, however rapid we make our breathing, we perceive no difference in the ease with which successive acts of inspiration are performed. This circumstance is a proof the most decisive, that the fibres of the bronchi have no independent rhythmical motions of contraction; and if we suppose them to contract only after the tubes have been expanded, we attribute to them an office for which simple elasticity would be perfectly adequate.

The idea of spasm of the bronchi was suggested to Cullen, and has been generally adopted, from inability to explain in any other way the symptoms of asthma. A little consideration, however, is sufficient to show the improbable nature of this supposition. The large bronchial tubes, and, in man, those even of the fourth and fifth ramifications, cannot be closed by reason of the cartilaginous rings or plates, which wholly or partially surround them. Supposing, then, the circular fibres to be muscular, only very small bronchial tubes could be closed by their action; and the closing of a few of

those tubes would only obstruct the passage of air to the small portions of lung to which they lead, and would not cause much difficulty of breathing. The spasm, to explain the symptoms of asthma, must be supposed to affect the small bronchial tubes in a considerable portion of the lungs; and as, in almost all cases of asthma, some shortness of breathing remains, in the intervals of the fits, we must admit, and, in fact, most physicians who have written on the subject have admitted, that some degree of spasm is permanent.

The necessity of such a supposition has, in great measure, ceased, in consequence of modern discoveries in morbid anatomy. Corvisart first pointed out diseases of the heart and large vessels as an occasional cause of fits of dyspnœa, formerly regarded as nervous, and confounded under the name of asthma. Laennec, and more recently Louis, have shown that emphysema of the lungs is the most common cause of this group of symptoms, and that the physical signs of dilatation of the air-cells may be discovered during life in most persons who present the symptoms of asthma.

Many of these persons can vary the capacity of their chests to a degree only just sufficient to supply them with the requisite quantity of oxygen, in favourable circumstances. Whenever their circulation is quickened, by exciting passions or by exercise,—or their power of expanding the chest is a little diminished, by the obstacle which a distended stomach offers to the descent of the diaphragm,—

or the air is prevented from freely entering the air-cells, in consequence of secretions in the bronchial tubes,—or the proportion of oxygen in a given volume of air is diminished; whether by increased temperature, as in heated apartments, or by diminished pressure, as in elevated situations, and in those states of the atmosphere which precede storms:—in fact, whenever, from any cause, their need of oxygen increases, or their means of inhaling it diminishes, these persons experience difficulty of breathing, or a fit of asthma.

There still, however, remain some cases, which at present we can only explain by supposing the dyspnœa to be nervous. It seems probable that the number of such cases will be still further diminished, and that many of those fits of asthma, which we are now forced to consider nervous, will be discovered to depend on some organic change which has as yet escaped our observation, perhaps on some morbid condition of the blood itself.

In fits of asthma really nervous, the difficulty of breathing must result from spasm, or from suspension of the normal action of the diaphragm and other muscles of inspiration.